I was born three years after the end of the civil war that led to the partitioning of Ireland. Although there was little violence in Belfast, it was a deeply divided city. Those at the top of the social heap were generally Protestant, and those at the bottom were Catholic. There was widespread discrimination in respect both of public housing and employment.

Although the term had not been invented, ethnic cleansing—in the sense of people of one religious persuasion being forced out of a neighbourhood by intimidation or arson by those of another persuasion—occurred sporadically. Underlying all this was fear by the party on top that their privileged position might be swept away by a differential birth rate or immigration. At the other end of the social scale people felt deep frustration and anger that they had been excluded from opportunity. Over this scene presided a parliament with constitutional machinery which for 60 years gave little encouragement to the involvement of the political minority in government. Although the bitter fruits of this situation have led to organised violence in the form of terrorism rather than genocide, the underlying social and political causes have points in common.

I conclude that genocide is the final stage in a three stage deterioration in social relationships. Imagine a society in which one group (A) is in control—usually but not always in the majority—and another (group B) regards itself or is regarded as the underdog and is usually in the minority. In addition to socioeconomic differences, group A is ethnically different from B in terms of language, skin colour, or social history. Group A perceives Group B as a present or future challenge, not only to its superior social and economic position but to its cultural identity. This leads to a sense of insecurity with fear for the future. On the other hand, group B perceives itself as excluded from social and economic opportunity and the right to express its cultural heritage, which leads to frustration and anger.

The resultant first stage of social deterioration is so common that few countries in the world can claim complete immunity from it. There is discrimination on a personal or group basis against group B by group A, often including some degree of segregation. There is also intimidation, with conscious or unconscious fostering of prejudice in private attitudes and the mass media. The second stage involves sporadic, often cyclical, unplanned violence including shop smashing, looting, arson, and riots. Fortunately, in most countries this is as far as such situations deteriorate.

The final and dreadful step, which leads to attempted genocide, involves a crucial additional factor. This is the active participation, either openly or in secret, of the state itself. It goes without saying that this also implies the involvement of individual politicians. In the 20th century the state may become involved not only through using the police and armed forces to seek out and destroy the group in question, but by misusing the records of the social welfare system to identify them. The mass media are often a crucial factor, manipulated by politicians to inflame public opinion by, for example, fanning tribal memories of long past victories and defeats. But we must not make the mistake of placing all the blame on politicians, for no act of genocide—whether in Auschwitz or Srebrenica—has taken place without a substantial measure of public consent.

So what of the role of the medical profession? Worldwide it can exercise an important influence in the prevention of genocide. It should insist that episodes of genocide are fully exposed, documented, and punished. But it should also use its powerful potential in advocacy to support the United Nations charter, the Helsinki Declaration on Human Rights, and all public policies that work to reduce social or political discrimination on the basis of race, colour, or religion whether by education, legislation, or other means.

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Reducing paracetamol overdoses

More likely to succeed through public education than package labelling

Medicines containing paracetamol (acetaminophen) are the commonest cause of intentional drug overdose in Britain[1-4] and are increasingly implicated elsewhere in Europe[3] and the United States. Around 70 000 cases of paracetamol overdose occur annually in Britain, and numbers are growing.[4] Fortunately, death from overdose is rare—about 200 cases a year in Britain—against a remarkable safety record of 30 million packs containing paracetamol sold each year.[1-4] Hospital resources have been concentrated on trying to anticipate the few patients most likely to develop serious liver injury after overdose[5 6] since many deaths are preventable by early intervention, including treatment with the antidote N-acetylcysteine.[5 6] A bigger problem, however, is how to predict who is going to take an overdose and how to stop them.

Last month, Britain's Medicines Control Agency launched a programme of consultation on the availability of solid dose analgesics, including paracetamol.[7] The aim is to reinforce the safe use of paracetamol. The programme proposes the inclusion of warnings about overdose in the product information statements, including the risk of serious liver damage from overdose and the need to seek immediate medical advice even if well. Current statements must declare the presence of paracetamol and include warnings not to exceed the stated dose and to consult a doctor if symptoms persist.

The Medicines Control Agency is also seeking views on limiting individual general sales of products containing paracetamol to 6 g for adults and 1.44 g for children—typically 12 tablets or capsules—and for pharmacy sales not to exceed 30 and 100 tablets for short and long term conditions respectively.[7] In announcing the programme of consultation, Gerald Malone, minister of health, stated that "the way forward is to ensure that full and accurate information reaches consumers, that information should be conveyed both on the label and patient leaflet, in a pack whose size meets their needs without leaving large numbers in the bathroom cabinet."

The success of these proposals, if they are carried, will depend largely on their success in deterring those most likely to take overdoses. The difficulty lies in reaching this target population. Most overdoses in Britain seem to result from impulsive acts.[1-4] Those who seek manipulative gain, especially adolescents, favour paracetamol for self poisoning. Paracetamol has replaced other drugs, such as aspirin, as a popular

choice for pain relief and, consequently, for overdose because selection is dictated by ready availability, sales, and prescription rates.[2 8 9] Studies from Oxford show that, although most people who overdose know that paracetamol can be dangerous, prior knowledge of potential death and warnings on labels, however shocking, were deterrents in only a quarter of patients interviewed.[1 2 4]

Restricting individual sales should affect the number of serious overdoses, although this is not entirely predictable.[9] In France, where packet size is limited to 8 g, overdose is common but severe hepatotoxicity and death are rare.[10] However, of people in Oxford who took more than 12 tablets, only 37% said that they would have taken smaller quantities had their packets been limited to 12 tablets.[4] The effect of blister packs is also uncertain. Although people using them were less likely than people using loose supplies to take more than 25 tablets (40% compared to 60%), the amount ingested reflected immediate availability.[4] The ever increasing number of formulations containing paracetamol poses practical problems for deterrent packaging, and, anyway, the new proposals on limiting pack size do not extend to effervescent tablets, granules, and sachets.[7]

Recommendations to limit the availability of designated drugs by reducing pack size or making larger amounts available only on prescription may reduce the number of serious overdoses. But given the impulsive nature of overdose, they are unlikely to reduce the total number of overdoses. The extra investment costs, if passed to the consumer, would seem an unfair financial penalty for the millions who heed recommended doses. Furthermore, selective restrictions could favour overdose with the next most available drug, or "cocktails" of unknown formulations and inferior safety record to that of paracetamol.

While we endorse the need to encourage consumers to read and heed drug safety information, we remain unconvinced, as do others,[4] about the likely impact of any form of warnings on labels and packets as a deterrent to overdose, especially among those who seek manipulative gain. We recommend disseminating public safety information about all the popular drugs through educational programmes in schools and via additional media such as television, radio, newspapers, and even the Internet. Information should encourage early hospital treatment after overdose, emphasising for paracetamol the high efficacy of the antidote.[6] Any campaign that publicises the toxicity of specific drugs should be monitored carefully to watch for increases in selection of other drugs.

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Comments on the proposals (reference MLX231) should reach Dr John Price, Department of Health, Medicines Control Agency, Room 1106 Market Towers, 1 Nine Elms Lane, London SW8 5NQ, before 10 January 1997 to allow implementation of any changes by April 1997.

Thromboembolism in primary pulmonary hypertension

No clear increased risk from oral contraceptives

Premature death in patients with clinical evidence of severe right ventricular strain and no obvious predisposing condition has been recognised for over a century. In 1952 Paul Wood distinguished this syndrome of primary pulmonary hypertension from the pulmonary vascular disease that complicates some congenital heart lesions,[1] recognising the very poor prognosis that continues to characterise this rare disorder. Its yearly incidence is between 1/200 000 and 1/1000 000, with the result that progress in understanding its pathology and interrupting its natural course has been slow. The overall mean survival remains under five years despite a variety of new treatments.

As in many systemic arterial diseases, there is increasing evidence that primary pulmonary hypertension is an endothelial disorder. The characteristic vascular obliteration that is observed as the condition progresses is thought to be the end stage of intense vasospasm and thrombosis, in different proportions in the predominantly "thromboembolic" and predominantly "plexogenic" histological types. The distinction between these as pathological or clinical entities is blurred, and it is likely that disruption of the antithrombotic and vasodilator functions of the pulmonary endothelium leads to irreversible structural pulmonary vascular disease. The endothelial products that are discussed most widely in this context are nitric oxide and prostacyclin, both of which are vasodilators and antithrombotic agents, and endothelin-1, a powerful vasoconstrictor found in raised concentrations in the serum of the fawn hooded rat, the only animal model for the disease. Prostacyclin activity is low in patients with primary pulmonary hypertension,[2] but abnormalities in the basal levels of nitric oxide or endothelin-1 are less distinct.

There is no agreement as to the importance of thromboembolism, as opposed to local thrombosis in situ, in this condition. A proportion of patients have evidence of recurrent thromboembolism, and this subset can now be recognised from the characteristic appearance on a radionuclide perfusion scan.[3] In patients without recognised recurrent emboli there is often histological evidence of microvascular thrombosis,[4] and in addition there may be clot in the central pulmonary arteries that does not interfere with blood flow.[5] The origin of such small and large vessel clots is not clear, but their occurrence supports the routine use of anticoagulants in all patients. Long term anticoagulation was shown to be associated with increased survival in the first large clinical series reported from the Mayo clinic in 1984.[6]

Most patients are women of childbearing age, and presentation during or after pregnancy is common. In established disease pregnancy is associated with rapid clinical deterioration.